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AVIATION PHYSIOLOGISTS BULLETIN

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CARBON MONOXIDE IN AIRCRAFT

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The air breathed by personnel in airplanes is ordinarily free from dangerous quantities of carbon monoxide. There are nevertheless several sources of carbon monoxide in military aircraft and under exceptional conditions the cockpit air may be contaminated with dangerous quantities of this gas. The exhaust fumes from the engines contain about 2% of CO. Serious poisoning will result from continuous exposure to one part of these fumes mixed with every 100 parts of air ventilating the cabin (o. g. 0.02%). Prolonged exposure to smaller concentrations may result in a loss of efficiency and present Army Air Force standards require that the concentrations of CO in the inspired air shall not exceed 0.005%. In many types of aircraft the hot exhaust gases are utilized indirectly to heat the cabin. The gases are led over pipes through which heat is transferred to air supplying the cockpit. Openings in the heat exchange system which result from defective materials, old age or bullet holes will lead to contamination of the heated air with carbon monoxide. Many medium and large airplanes are now being equipped with auxiliary gasoline powered generator units or with gasoline powered heating and ventilating systems. Bullet holes or defects in the exhaust lines of any of these units will lead to contamination of the cabin air with CO. In view of the hazard of CO poisoning under such circumstances a knowledge of the physiological factors involved in carbon monoxide poisoning has become important to the aviation physiologist and to the airplane manufacturer.

The poisonous effects of CO are a result of its action on the transport of oxygen in the blood. Hemoglobin combines with CO in much the same way as it combines with oxygen. Thus the carbon monoxide dissociation curve of hemoglobin is similar to that of oxygen, and it is affected by changes of pH in the same way as the oxygen dissociation curve. However, the affinity of hemoglobin for CO is some 200 fold its affinity for oxygen so that small concentrations of CO readily displace oxygen from the blood. It was early discovered by

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Haldane that in blood equilibrated with mixtures of oxygen and carbon monoxide the ratio of carboxy- to oxyhemoglobin is proportional to the ratio of the partial pressures of the two gases. In human blood the proportionality constant is 210 so that blood exposed to 210 mm. Hg of oxygen and 1 mm. of CO is equally saturated with oxygen and CO. Haldane's Law may be extended to conditions in which reduced hemoglobin is also present and the saturations of oxy-, reduced, and carboxyhemoglobin may be predicted from the composition of the gas phase in equilibrium with the blood.

Since carbon monoxide displaces oxygen from hemoglobin, its poisonous action may be considered as a form of anoxia. Symptoms common to both oxygen want and to acute CO-poisoning are a rapid pulse rate, disturbance of vision, inability to concentrate and cold sweating of the palms and forehead. One or more of these symptoms are generally noticeable subjectively before the onset of collapse. Moderate CO poisoning is usually less noticeable subjectively than an equivalent reduction of oxyhemoglobin resulting from low oxygen pressures. This may be because the partial pressure of oxygen in arterial blood is unaffected by the presence of carboxyhemoglobin, and a normal pressure of oxygen (but not a normal oxygen saturation) is supplied to the arteriolar end of the tissue circulation. This hypothesis agrees with the observation that the chemoreceptors in the carotid bodies are insensitive to the presence of carboxyhemoglobin in the arterial blood whereas they do respond to low oxygen tensions. A notable difference between the two forms of anoxia is the absence of cyanosis during CO poisoning. This is because the absorption spectrum of carboxyhemoglobin is not greatly different from that of oxyhemoglobin in the visible range. The effects of moderate CO poisoning, are not permanent. However, several hours are required to eliminate CO from the blood so that in contrast to anoxia produced by decreased oxygen pressure the symptoms remain for a considerable time after the exposure. Headaches frequently develop over such long periods.

The exchange of carbon monoxide between the blood and the alveolar air takes place slowly. This is a result of the small partial pressure of CO which sets a limit to the pressure gradient available for diffusion across the alveolar membranes. The rate of uptake or of elimination of CO from the blood conforms approximately to the exponential form predicted by diffusion theory; initially the rate is large but it diminishes as equilibrium is approached. Four or five hours are generally required to reach equilibrium although a dangerous quantity of CO may enter the blood in a few minutes if the concentration in the inspired air is sufficiently great. Figure I shows the times required to reach a dangerous concentration of CO in the blood (enough to reduce the oxygen content of the blood to 17.0 volumes percent) as a function of altitude and percentage CO in the inspired air. The rates of uptake or of elimination are approximately proportional to the ventilation rate so that moderate exercise involving a doubling of the ventilation rate will halve the times given in Figure I.

Figure I also illustrates the effects of altitude on the ability to tolerate carbon monoxide. As may be expected from the foregoing considerations, the effects of any given degree of CO poisoning are greatly accentuated by additional anoxia brought about by altitudes. For example, 20% saturation of the blood with carbon monoxide is just perceptible by experienced individuals at sea-level; at 12,000 feet, however, where the oxygen saturation is decreased by a further 20%, most individuals would be on the verge of collapse. Serious symptoms have been observed at 7,000 feet under these conditions.

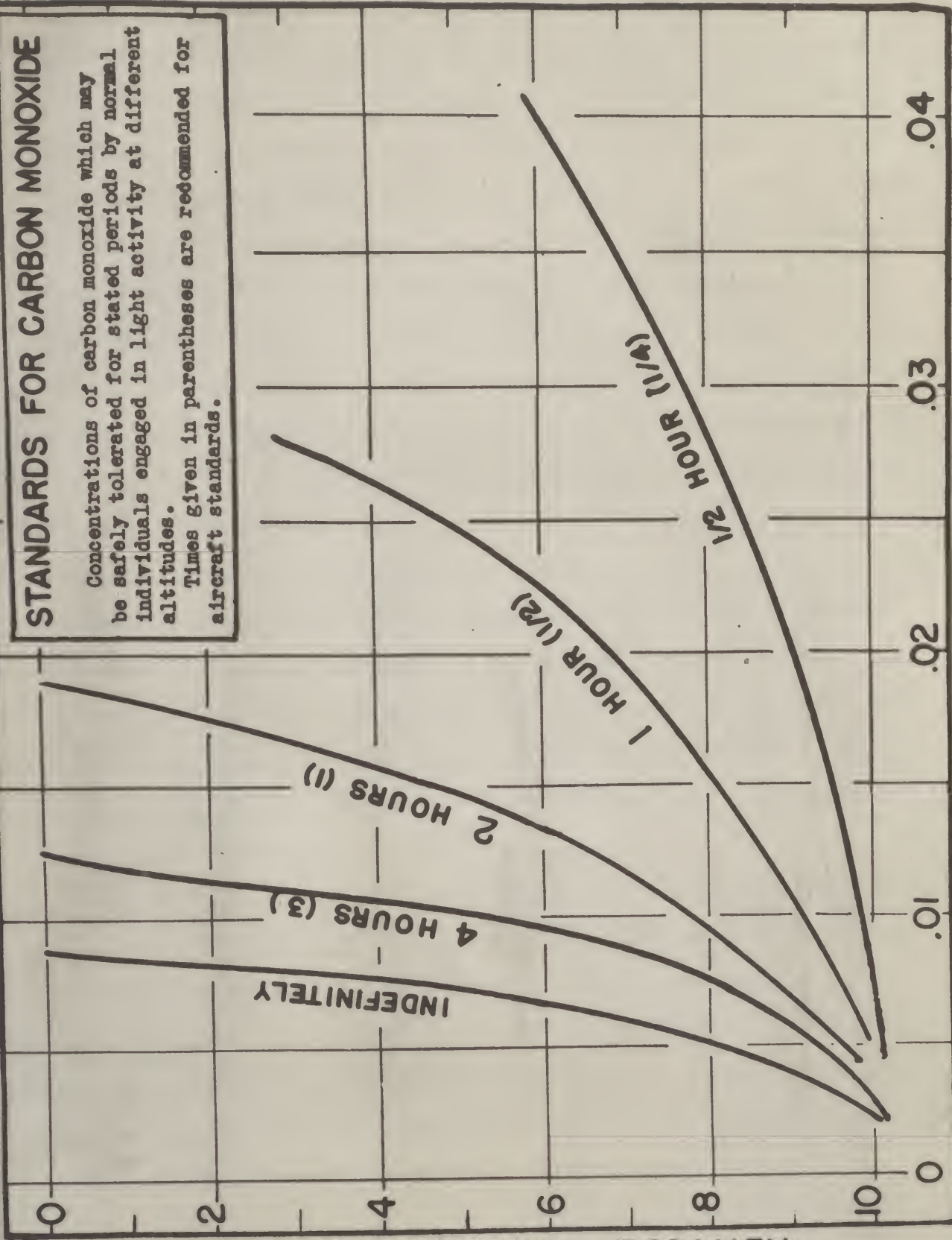
The harmful effects of CO are greatly diminished as a result of breathing pure oxygen. Oxygen alleviates the symptoms in two ways - 1) more oxygen is transported to the brain in the form of physical solution. This effect occurs within a few seconds and may result in dramatic recovery from severe symptoms of CO poisoning; 2) pure oxygen displaces CO from hemoglobin according to Haldane's Law and hence increases its rate of elimination from the blood.

STANDARDS FOR CARBON MONOXIDE

Concentrations of carbon monoxide which may be safely tolerated for stated periods by normal individuals engaged in light activity at different altitudes.

Times given in parentheses are recommended for aircraft standards.

ALTITUDE - THOUSANDS OF FEET



PERCENTAGE CARBON MONOXIDE IN INSPIRED AIR

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The most direct measure of the degree of carbon monoxide poisoning is the concentration of carbon monoxide in the blood. A simple and ingenious gasometric method for the analysis of CO in small amounts of blood has been developed by Scholander and Roughton. The blood sample and reagents are drawn into a 1 cc. tuberculin syringe through a graduated length of capillary tubing which is attached to the syringe in place of the needle. The hemoglobin is converted to methemoglobin with ferricyanide, thus liberating combined oxygen and CO. A bubble of CO_2 is then formed in the syringe from acid and bicarbonate. The volume of the bubble is large compared to the volume of the blood gases so that the partial pressure of oxygen, CO and nitrogen within the bubble is very small. The bubble of CO_2 thus replaces the Torricellian vacuum which is ordinarily used to extract gases from blood. After the gases have diffused into the bubble, the CO_2 and oxygen are absorbed with pyrogallol. The remaining gas, consisting of CO and nitrogen, is then forced into the graduated capillary where its volume is measured before and after absorption of the CO with Winkler's solution. The analysis requires only 40 cu. mm. of blood which may be conveniently obtained from a single finger-prick. The error of the method is $\pm 2\%$ saturation -- an accuracy which compares favorably with that of other more elaborate gasometric or spectrophotometric methods.

The analysis of CO in air is valuable for the detection and location of sources of CO. The instrument most commonly used for flight testing depends upon the oxidation of CO to CO_2 . This reaction occurs at room temperature over a catalyst (hopcalite) with the liberation of 31,000 kilocalories per mol. The heat so liberated is usually measured with thermopiles and the instrument arranged to give a continuous record of the CO concentration. Several chemical methods are available for the determination of CO in aliquot samples.

John R. Fappenheimer

AVIATION PHYSIOLOGISTS

Since the first issue of the Bulletin, three more classes of Aviation Physiologists have been graduated from the School of Aviation Medicine, Randolph Field. The graduates and their present assignments are as follows.

Class VII, Beginning March 15, 1943

<u>Name and Rank</u>	<u>Degree</u>	<u>Station</u>
Alexander, Stephen J., Jr., 1st Lt., M.C.	M.D. 1941, Cincinnati	Las Vegas AAF, Nevada
Barnes, Russell G., Jr. 1st Lt., M.C.	M.D. 1939, Minnesota	Greenville AAB South Carolina
Blair, Albert P., 1st Lt., A.C.	Ph.D. 1940, Indiana	Pyote AAF, Texas
Brown, Gordon A., 1st Lt., M.C.	M.D. 1941, Minnesota	Maxwell Field, Alabama
Browne, William C., 1st Lt., M.C.	M.D. 1941, Michigan	Maxwell Field, Alabama
Cahoon, Daniel H., 1st Lt., M.C.	M.D. 1941, Chicago	S. A. M. Randolph Field, Texas
Dautrich, Albert W., 1st Lt., M.C.	M.D. 1939, Yale	Tyndall Field, Florida
Fowler, Ward S., 1st Lt., M.C.	M.D. 1941, Harvard	MacDill Field, Florida
Hartmann, William L., 1st Lt., M.C.	M.D. 1939, Johns Hopkins	Dale Mabry Field, Florida
Heath, Erle M., 1st Lt., M.C.	M.D. 1940, Pittsburgh	Moses Lake AAF Washington
Keiber, Henry F., 1st Lt., M.C.	M.D. 1938, Long Island	San Antonio Avia- tion Cadet Center Texas
Martin, Archibald G.M., III, 1st Lt., M.C.	M.D. 1940, Duke	Harlingen AAF, Texas
Mazer, Milton, 1st Lt., M.C.	M.D. 1935, Pennsylvania	Laredo AAF, Texas
McRae, Donald Roswell Jr., 1st Lt., M.C.	M.D. 1941, Georgia	San Antonio Aviation Cadet Center, Texas
Mendenhall, Norman E., 1st Lt., M.C.	M.D. 1938, Pennsylvania	Avon Park Bombing Range, Florida
Streett, James C., Jr., 1st Lt., A.C.	Ph.D. 1939, Princeton	Santa Ana AAB, California
Chapanis, Alphonse, 2nd Lt., A.C.	Ph.D. 1943, Yale	Aero Medical Laboratory, Ohio
Colwin, Arthur L., 2nd Lt., A.C.	Ph.D. 1936, McGill	Davis-Monthan Field Arizona
Forsyth, John W., 2nd Lt., A.C.	Ph.D. 1941, Princeton	Columbia AAB South Carolina
Schoenborn, Henry W., 2nd Lt., A.C.	Ph.D. 1939, New York	March Field, California

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<u>Name and Rank</u>	<u>Degree</u>	<u>Station</u>
Thomson, John D., 2nd Lt., A.C.	Ph.D. 1940, Iowa	Selfridge Field, Michigan
Class VIII, Beginning April 26, 1943		
Gaxiola, Marco A., Major, M.C.		Mexican Army
Motley, Hurley L., Capt., M.C.	M.D. 1936, Harvard	Maxwell Field, Alabama
Ney, Joseph, Capt., M.C.	M.D. 1935, Harvard	San Antonio Aviation Cadet Center, Texas
Ahrens, Edward H., 1st Lt., M.C.	M.D. 1941, Harvard	Eglin Field, Florida
Goodfriend, James, 1st Lt., M.C.	M.D. 1940, Northwestern	School of Applied Tactics, Florida
Leo, Sidney D., 1st Lt., M.C.	M.D. 1936, Basel, Switz.	Moses Lake AAF, Washington
Mahady, Stephen C. F., 1st Lt., M.C.	M.D. 1939, Harvard	S. A. M. Randolph Field, Texas
McNeil, Robert J., 1st Lt., M.C.	M.D. 1939, Creighton	Santa Ana AAB, California
Riesen, Austin H., 1st Lt., A.C.	Ph.D. 1939, Yale	Greenville AAB South Carolina
Young, Dennison, 1st Lt., M.C.	M.D. 1938, Tufts	Dale Mabry Field, Florida
Warner, Eldon D., 2nd Lt., A.C.	Ph.D. 1941, Wisconsin	Hamilton Field, California
Wilson, Charles C., 2nd Lt., A.C.	Ph.D. 1943, Duke	Columbia AAB, South Carolina

Class IX, Beginning June 7, 1943

Duimstra, Fred, Capt., M.C.	M.D. 1937, Arkansas	San Antonio Aviation Cadet Center, Texas
Krieger, Herbert D., Capt., M.C.	M.D. 1935, Wisconsin	Maxwell Field, Alabama
Orndorff, John R., Capt., M.C.	M.D. 1935, Northwestern	McChord Field, Washington
Goodman, Seaburt, 1st Lt., M.C.	M.D. 1940, Toronto	Buckingham Field, Florida
Grabber, Harold L., 1st Lt., M.C.	M.D. 1941, Kansas	San Antonio Aviation Cadet Center, Texas
Hallenbeck, G. A., 1st Lt., M.C.	M.D. 1940, Northwestern	Aero Medical Laboratory, Ohio
Palmeri, Vincent J., 1st Lt., M.C.	M.D. 1935, St. Louis	Santa Ana AAB, California
Rodgers, Bradford D., 1st Lt., M.C.	M.D. 1941, Cincinnati	Kingman AAF, Arizona
Smith, Sidney, 1st Lt., M.C.	M.D. 1941, Rush	Buckley Field, Colorado

<u>Name and Rank</u>	<u>Degree</u>	<u>Station</u>
Straus, Oliver H., 1st Lt., M.C.	M.D. 1940, Columbia	Mountain Home AAF, Idaho
Titelbaum, Sydney, 1st Lt., A.C.	Ph.D. 1938, Chicago	Westover Field, Massachusetts
Witherbec, Orville O., 1st Lt., M.C.	M.D. 1935, S. California	Maxwell Field, Alabama
Castor, John G., 2nd Lt., F.A.	Ph.D. 1940, Harvard	Barksdale Field, Louisiana
Foulks, James G., 2nd Lt., A.C.	Ph.D. 1943, Johns Hopkins	Bolling Field, D.C.
Sonnenblick, Benjamin P., 2nd Lt., A.C.	Ph.D. 1938, N. Y. U.	Clovis AAF, New Mexico
Warner, Edward N., 2nd Lt., A.C.	Ph.D. 1940, Ohio State	Dale Mabry, Florida

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CONTROL OF INCAPACITATING EFFECTS OF DECOMPRESSION

II. Denitrogenation before Ascent

The incidence of bends and chokes can be reduced or eliminated by removal of nitrogen from the body by breathing pure oxygen before flight. Such denitrogenation can be employed either as a substitute for selection procedures or as an accessory method used in conjunction with selection to make the incidence of decompression sickness a negligible factor in high altitude operations.

The time required for denitrogenation before ascent in order to effect protection from decompression sickness varies from individual to individual. About four hours are needed to protect the more susceptible subjects during exposures to 38,000 feet, but the evidence at hand shows that 30 to 45 minutes of preliminary nitrogen removal produces a significant degree of protection in many individuals. This protection is indicated by the increased time that the individual can remain at altitude before the onset of symptoms, the protection being essentially complete for some men.

When the period of denitrogenation is limited to an hour or so, it is considered important to exclude mask leaks, so that the nitrogen tension in the

Inspired gas is reduced essentially to zero, and to be sure that no air is inspired at any time until all flying above 30,000 feet is completed. If any air is breathed, some nitrogen will be regained and the effectiveness of the denitrogenation reduced. After very thorough denitrogenation by long periods of pure oxygen inhalation (e. g. eight hours), it may be permissible to breathe air for a few minutes, a convenience in transferring from denitrogen station to plane. But it is at present recommended that this should not be done after the shorter, more practicable denitrogenation procedures.

There is conflicting evidence as to whether exercising in order to stimulate the respiration and circulation during short denitrogenation periods increases the degree of protection. In all denitrogenation procedures it is generally agreed, however, that rest is important during the last fifteen minutes before flight, a finding which may be related to the fact that exercise after reaching altitude tends to precipitate bends.

The protection afforded by denitrogenation is believed to be due to a reduced tendency for the formation and growth within the body of the gas bubbles which are generally supposed to cause decompression sickness. In agreement with this assumption is the observation that fewer gas bubbles appear within the veins of experimental animals on exposure to reduced pressure if they are allowed to breathe 100% oxygen for a while before decompression. The occurrence of such gas bubbles extravascularly as well as intravascularly has long since been demonstrated, and it is assumed that symptoms of decompression sickness result if the bubbles grow to sufficient size in certain critical locations.

The development of symptoms within a given length of time at altitude is therefore believed to depend upon the existence of suitable conditions for (1) the initial formation of bubbles and (2) their sufficiently rapid growth. Recent theoretical studies and in vitro experiments have developed concepts concerning the factors which determine both the formation and the rate of growth.

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To start the formation of bubbles it is necessary that the tissues be supersaturated with gas. Such a state of supersaturation exists whenever the sum of the partial pressures of all dissolved gases (including water vapor) exceeds the total pressure upon the tissues. In addition suitable "nuclei" for bubble formation must be present. The exact nature of the "nuclei" and their distribution within the tissues is not understood, but their importance can be demonstrated in vitro where clean solutions may remain supersaturated for hours, but gas bubbles rapidly form if any dust particles are present. The bubbles contain a mixture of the various gases present in solution.

Once a small bubble is formed its rate of volume increase during aircraft ascent depends upon (1) the rate at which it expands because of the decreasing atmospheric pressure, plus (2) the rate of transfer of additional molecules from the tissue fluids to the gas phase. After leveling off, the rate of bubble growth is determined by the second factor only. This rate of transfer of gas molecules to a bubble increases with the diffusion coefficients of the various gases as well as with the degree of supersaturation as determined by the concentrations of the gases and the atmospheric pressure. The concentrations at any instant are in turn determined by the initial concentrations at the start of the ascent, the amount of gas already transferred to gas bubbles, and the rate of removal during the ascent by the circulation and by metabolic processes.

In the light of these concepts denitrogenation by breathing pure oxygen is believed to reduce bubble formation and growth by reducing the tendency for development of a state of supersaturation. In the first place the sum of the partial pressures of the dissolved gases present in the tissues is reduced before ascent. Oxygen inhalation of course does not alter the sum of the partial pressures in the lungs nor presumably in arterial blood. There is, however, an enormous drop in oxygen tension as the blood traverses the capillaries, where about the same amount of oxygen must be removed from each cc. of blood as normally.

This oxygen tension drop is much larger than when air is being breathed because the tension is reduced very rapidly as oxygen is withdrawn starting at the high arterial tensions attained when inspiring 100% oxygen at sea level. For this reason when pure oxygen is breathed instead of air the increase in oxygen tension is much less in metabolizing tissues than the reduction in nitrogen tension, and consequently the sum of the partial pressures of the dissolved gases becomes much lower in the tissue fluids. Not only is the total tissue gas tension thus initially lower after breathing pure oxygen, but during ascent it can fall more rapidly than when nitrogen is present. This is at least in part because the oxygen is continually being removed by cellular metabolism, whereas the only means of removing nitrogen is by diffusion into the capillaries when nitrogen tension in arterial blood is lowered. Thus denitrogenation by inspiring 100% oxygen acts to prevent the symptoms of decompression sickness both by lowering the total tensions of the gases in tissues before take-off and by accelerating their removal during ascent. Consequently a higher altitude must be reached before a state of supersaturation is developed.

Though we seem to be well on the way, through selection and denitrogenation, to a possible solution of the "bends" problem in high altitude flying, the chain of events occurring between the formation of gas bubbles and the initiation of nerve impulses in pain fibers is still obscure. Research along these lines may reveal other ways of controlling or preventing these incapacitating effects of low pressure. For example, when the origin of the daily variation in an individual's susceptibility is more clearly defined, we will know whether it is possible to predict by some test his performance on any one day.

Frank Brink, Jr. and

M. G. Larrabee

SUGGESTIONS FROM THE ALTITUDE TRAINING UNITS

1. By inclosing the moving picture projector in a box with a glass opening in front and one side removable for adjustment of the machine, a satisfactory soundproofing of the projector itself has been obtained at Colorado Springs.
2. Units using the Hytor Pump may get into difficulty when the current is shut off during a run or when the motor stops for any other reason. The pump can not usually be started against the load and the suction may pull water into the chamber when attempts are made at starting. Lieutenants Copeland and Zeiner have eliminated this problem by placing a by-pass valve between the chamber operator's valve and the suction pump. When the motor stops all valves on chamber and lock are closed, the new valve opened and the motor may be easily started when the current comes on again.
3. When a bolt must be set in a spot where concrete has been chipped out, melted sulphur may be poured into the hole to anchor the bolt in place.
KEEP YOUR GAS MASK ON WHILE THIS IS BEING DONE.

Lieutenants Copeland & Zeiner
Peterson Field, Colorado Springs

1. Metal cots found in Station Hospitals can have castors set in by removal of metal caps found on the end of the legs. If two 8" pieces of $1\frac{1}{2}$ " angle iron are welded onto the bed (parallel to the long axis of the bed, 6" above the castor), a solid shelf can be made. On the shelf can now be placed a chest containing all First Aid equipment and emergency drugs and instruments. The shelf will also have room for a high pressure walk-around cylinder assembly. Thus we have an emergency cot which may be left in the supply room until needed, rolled to the lock for use and upon arrival contain everything which might be needed for care of the patient. So that the cot can never fold up while being rolled, the safety catches are welded in position. Room still remains for a

small tank of carbon dioxide - oxygen mixture. If desirable, the patient may now be rolled to another room to prevent his being disturbed by questioning or noise.

2. Changes in the policy of film distribution have caused difficulty in obtaining copies of Training Film 1-313. The correct method of obtaining a copy under the present set-up is by writing a letter to the Chief of Technical Data Branch, Air Force Section, Air Service Command, Patterson Field, Ohio.

Captain Dickson
Bolling Field, D. C.

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NOTES

1. The Air Surgeon has been informed that a new Military Occupational Specialty Altitude Chamber Technician has been established and designated as 617. This number will be used in reporting strength of enlisted chamber crews (Air Corps) on forms 127 and 128. The specialty number will be published in the forthcoming revision of AR615-26.

2. With the publication of the revision of AAF Regulation 35-12, Altitude Chamber Technicians will be authorized to wear the engineering sleeve patch. It was impossible to obtain a sleeve patch designed specifically for Altitude Chamber Technicians.